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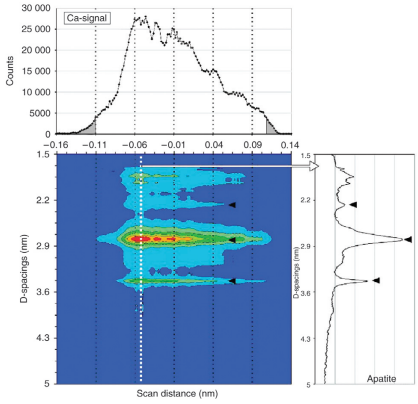
Apatite deposition in vascular calcification



There is increasing interest in vascular calcification in end-stage renal disease, as it has been demonstrated that it correlates with morbidity and mortality in this condition. The identity of the mineral that is deposited was studied in two animal models of this disease, one induced by adenine and a high-phosphate diet and the other induced by 5/6 nephrectomy in the presence of a high-phosphate diet and high-calcitriol treatment. Aortic sections were examined by synchrotron X-ray-μ-fluorescence and diffraction, by which all the minerals were examined. Atomic absorption was also used to estimate bulk calcium and magnesium. Three types of precipitates were seen: an amorphous precipitate, apatite, and a combination of apatite and magnesium-containing whitlockite. The occurrence of these precipitates differed significantly between the two models. Furthermore, the combination of apatite and whitlockite was found exclusively in the calcitriol-treated animals. Given that vitamin D stimulates not only calcium but also magnesium absorption in the intestine, these results suggest an explanation for the presence of whitlockite in these animals. The remarkable finding of these crystalline forms also raises the question of whether there are cells in the aortic wall capable of producing a crystalline precipitate, previously thought to occur largely in bone and in kidney stones. **See page 298.**

Cost of applying K/DOQI guidelines for phosphate control

In 2003, the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (K/DOQI) published Clinical Practice Guidelines for the treatment of bone metabolism and disease in chronic kidney disease. These guidelines advocated broad usage of non-calcium-based phosphate binders such as sevelamer. In this issue, a Canadian group estimates the cost of such treatment in a single dialysis unit and estimates what it would have cost to switch patients to sevelamer on the basis of the K/DOQI guidelines. Almost half of their 400-odd patients would qualify for the switch. The results are astounding. For this single hemodialysis unit, the cost of the switch was estimated to be about a half a million US dollars. Needless to say, a more compelling study would need to be designed before one could recommend wholesale switching from calcium-based phosphate binders to sevelamer. **See page 312.**

Daily hemodialysis and phosphate control

The control of calcium and phosphate homeostasis in end-stage kidney disease is one of the more difficult problems that face patients with this condition. As they report in this issue, Ayus *et al.* devised a controlled clinical trial in which they compared two groups of patients, one treated with daily hemodialysis for 3 hours per day 6 days a week and the other treated in a more conventional manner with three 4-hour sessions per week. Control of the serum phosphate was superior in those with daily dialysis after 1 year of treatment, as was the amount of phosphate removed during the dialysis. That group's use of phosphate binders was also reduced. Many more patients on daily dialysis achieved their targets in mineral metabolism, such as reduction of phosphate and improvement in calcium or in parathyroid hormone levels. Other metabolic parameters were also improved, such as the daily protein catabolic rate. These studies demonstrate a difficult problem for the renal health-care system. Daily dialysis (not surprisingly) is superior to conventional hemodialysis, which raises the question: Now what do we do? **See page 336.**

Proteinuria and gene expression

Proteinuria is now thought to be itself the culprit in mediating tubulointerstitial disease and eventual progression to renal failure. To analyze the potential mechanisms by which these events occur, Rudnicki *et al.* performed microarray analysis on small identified segments of proximal tubule removed by laser capture microdissection. Using as controls the normal sections of kidneys removed for renal-cell carcinoma, the authors compared the extracted RNA with that of proximal tubules microdissected from renal biopsies of patients with a variety of proteinuric states. Analysis of these microarrays showed 168 genes that were differentially expressed by the proteinuric proximal tubules. This included alterations in the Wnt signal-transduction pathways and upregulation of genes involved in cell proliferation and cell cycle control, cell differentiation, immune response, intracellular transport, and metabolism. Although these findings showed some expected results, such as upregulation of profibrotic genes, they also showed that there was an increase in genes thought to be protective of tubules, such as BMP-7, providing a complex picture of disease-causing genes being upregulated simultaneously with those that promote healing. **See page 325.**